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Evaluation of Serum Amyloid A as a Prognostic Marker in Community-Acquired Pneumonia

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Abstract: ***Background***: Community-acquired pneumonia (CAP) remains a significant cause of morbidity and mortality worldwide. This study evaluated the prognostic value of Serum Amyloid A (SAA) in CAP patients and compared it with established biomarkers and severity scores.

Methods: In this prospective, observational cohort study, we enrolled 200 adult patients admitted with CAP at a tertiary care hospital over 24 months. SAA, C-reactive protein (CRP), and procalcitonin (PCT) levels were measured on admission. The primary outcome was 30-day all-cause mortality. Receiver operating characteristic (ROC) analysis, logistic regression, and net reclassification improvement (NRI) were performed to assess SAA's prognostic value.

Results: The 30-day mortality rate was 14% (28/200). SAA levels were significantly higher in non-survivors compared to survivors (median 389.7 vs 142.5 mg/L, p<0.001). ROC analysis showed SAA (AUC 0.86, 95% CI: 0.79-0.93) outperformed CRP (AUC 0.78) and PCT (AUC 0.81) in predicting mortality. An SAA level >275 mg/L independently predicted 30-day mortality (adjusted OR 3.85, 95% CI: 1.68-8.82, p=0.001) after adjusting for age, disease severity, and other biomarkers. Adding SAA to the Pneumonia Severity Index improved risk reclassification (NRI 0.21, p=0.001) and discrimination (IDI 0.056, p<0.001).

Conclusions: SAA demonstrates superior prognostic accuracy for 30-day mortality in CAP compared to traditional biomarkers and provides additional value when combined with existing severity scores. These findings suggest SAA could be a valuable tool for risk stratification in CAP, potentially improving clinical decision-making and patient outcomes.

Keywords: Community-acquired pneumonia, Prognostic biomarker, Mortality prediction, Risk stratification, Serum Amyloid A,

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INTRODUCTION

Community-acquired pneumonia (CAP) remains a significant global health concern, contributing substantially to morbidity and mortality worldwide. Despite advancements in medical care and antimicrobial therapies, CAP continues to pose challenges in terms of accurate diagnosis, prognosis, and management (Torres et al., 2013). The severity of CAP can vary widely, ranging from mild cases manageable with outpatient treatment to severe cases requiring intensive care unit (ICU) admission. This variability underscores the critical need for

reliable prognostic markers that can aid clinicians in risk stratification and guide appropriate treatment decisions. In recent years, there has been growing interest in the use of biomarkers to enhance the accuracy of CAP prognosis. While several biomarkers have been studied, including C-reactive protein (CRP) and procalcitonin (PCT), the search for more sensitive and specific markers continues (Christ-Crain and Müller, 2007). One promising candidate that has emerged is Serum Amyloid A (SAA), an acute-phase protein primarily produced by the liver in response to inflammatory stimuli.

SAA has garnered attention due to its rapid and significant increase during acute inflammation, often surpassing the magnitude of CRP elevation (Malle and De Beer, 1996). This characteristic suggests that SAA might offer enhanced sensitivity in detecting and monitoring inflammatory processes associated with CAP. Moreover, SAA has been implicated in various aspects of the immune response, including leukocyte recruitment and modulation of cytokine production, indicating its potential role in reflecting the overall inflammatory state in pneumonia patients (Eklund et al., 2012). The pathophysiology of CAP involves a complex interplay between microbial pathogens and the host immune response. Upon infection, the respiratory epithelium and alveolar macrophages initiate an inflammatory cascade, leading to the production of various cytokines and acute-phase proteins. SAA, as part of this acute-phase response, is rapidly synthesized and released into the circulation. Its levels have been shown to correlate with the severity of inflammation in various conditions, suggesting its potential utility in assessing CAP severity (Uhlar and Whitehead, 1999).

Previous studies have explored the role of SAA in respiratory infections, demonstrating its elevation in both viral and bacterial pneumonia. For instance, Nakayama et al. (2016) reported that SAA levels were significantly higher in patients with CAP compared to healthy controls and correlated with disease severity. Similarly, Tosanguan and Kitiyakara (2018) found that SAA levels on admission were predictive of adverse outcomes in CAP patients, including the need for mechanical ventilation and mortality. The potential advantages of SAA as a prognostic marker in CAP are multifaceted. First, its rapid response to inflammation could allow for earlier detection of severe cases, potentially enabling more timely intervention. Second, the magnitude of SAA elevation might provide a more nuanced assessment of disease severity compared to traditional markers. Third, the biological functions of SAA in immune modulation suggest that its levels might reflect not only the presence of inflammation but also the quality of the host response to infection.

However, despite these promising aspects, the utility of SAA as a prognostic marker in CAP has not been fully established. Questions remain regarding its specificity, optimal cut-off values for risk stratification, and its performance relative to or in combination with existing prognostic tools such as the Pneumonia Severity Index (PSI) or CURB-65 score (Lim et al., 2003; Fine et al., 1997). Additionally, the influence of factors such as age, comorbidities, and timing of measurement on SAA levels in the context of CAP requires further elucidation. The integration of biomarkers like SAA into clinical practice for CAP management holds the potential to improve patient outcomes through more accurate risk stratification and tailored treatment approaches. By identifying high-risk patients early, clinicians could make more informed decisions regarding the intensity of care, choice of antibiotics, and need for adjunctive therapies. Conversely, recognizing low-risk patients could facilitate safe outpatient management, reducing unnecessary hospitalizations and healthcare costs.

Furthermore, the evaluation of SAA as a prognostic marker in CAP aligns with the broader trend towards personalized medicine in infectious diseases. As our understanding of the host response to infection grows, biomarkers that reflect individual variations in this response become increasingly valuable. SAA, with its diverse roles in inflammation and immunity, could provide insights into the patient-specific aspects of CAP pathogenesis and prognosis. In light of these considerations, there is a clear need for comprehensive studies evaluating the prognostic value of SAA in CAP. Such research should aim to establish the relationship between SAA levels and clinically relevant outcomes, determine its performance characteristics in diverse patient populations, and assess its potential to enhance existing prognostic models. By addressing these questions, we can better define the role of SAA in CAP management and potentially improve the care

of patients with this common but potentially severe infection.

The aim of this study is to evaluate the prognostic value of Serum Amyloid A (SAA) in patients with community-acquired pneumonia (CAP) and to assess its potential as a biomarker for predicting disease severity, clinical outcomes, and mortality risk.

Methodology

Study Design and Setting: This prospective, observational cohort study was conducted at the United Institute of Medical Sciences, Prayagraj. The study duration was 24 months, from June 2021 to June 2023. This timeframe was chosen to account for potential seasonal variations in pneumonia incidence and to ensure a sufficient sample size for robust statistical analysis.

Study Population and Sampling: Consecutive adult patients (≥18 years old) admitted to the emergency department or medical wards with a diagnosis of CAP were recruited using a systematic sampling approach. The sample size was calculated using G*Power 3.1 software, assuming a moderate effect size (Cohen's d = 0.5), an alpha level of 0.05, and a power of 0.8. Based on these parameters and accounting for an estimated 15% dropout rate, the target sample size was determined to be 200 patients.

Inclusion and Exclusion Criteria:

Inclusion criteria:

1. Age ≥18 years
2. Clinical diagnosis of CAP based on the presence of at least two of the following symptoms: cough, sputum production, dyspnea, chest pain, or fever (>38°C)
3. Radiological evidence of new infiltrate on chest X-ray or CT scan consistent with pneumonia
4. Onset of symptoms in the community setting (i.e., not hospital-acquired)

Exclusion criteria:

1. Hospitalization within the previous 14 days to exclude healthcare-associated pneumonia
2. Immunocompromised status (e.g., HIV/AIDS with CD4 count <200 cells/μL, active malignancy undergoing chemotherapy, solid organ transplant recipients on immunosuppressive therapy)
3. Pregnancy or lactation
4. Chronic inflammatory conditions known to elevate SAA levels (e.g., rheumatoid arthritis, inflammatory bowel disease)
5. Severe liver disease (Child-Pugh class C) that might affect SAA production
6. Patients with do-not-resuscitate orders or those deemed unlikely to survive the next 24 hours due to comorbid conditions

Testing Methodology

Upon admission, demographic data, clinical history, and physical examination findings were recorded for all participants. Severity of pneumonia was assessed using the Pneumonia Severity Index (PSI) and CURB-65 score. Blood samples for SAA measurement were collected within 24 hours of admission, prior to the initiation of antibiotic therapy. SAA levels were measured using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Manufacturer, Country) according to the manufacturer's instructions. The assay had a detection range of 0.1-500 mg/L, with an intra-assay coefficient of variation (CV) <5% and an inter-assay CV <7%. All samples were analyzed in duplicate, and the mean value was used for statistical analysis.

In addition to SAA, other laboratory parameters were measured as part of routine clinical care, including complete blood count, C-reactive protein (CRP), procalcitonin (PCT), blood urea nitrogen, serum creatinine, and arterial blood gases. Microbiological investigations, including blood cultures and sputum cultures or urinary antigen tests for Streptococcus pneumoniae and Legionella pneumophila, were performed according to standard clinical protocols.

Patients were followed up for 30 days after admission or until hospital discharge, whichever came later. The primary outcome measure was 30-day all-cause mortality. Secondary outcomes included need for ICU admission, development of complications (e.g., pleural effusion, empyema, acute respiratory distress syndrome), length of hospital stay, and time to clinical stability (defined as normalization of vital signs and ability to maintain oral intake for 24 hours).

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Statistical Analysis

Statistical analysis was conducted using SPSS 25.0. Continuous variables were presented as means ± SD for normal distribution or medians with IQR for non-normal distribution. Categorical variables were expressed as frequencies and percentages. The Shapiro-Wilk test assessed normality. Group comparisons utilized Student's t-test or Mann-Whitney U test for continuous variables, and chi-square or Fisher's exact test for categorical variables. ROC curve analysis evaluated SAA's predictive power for 30-day mortality and other outcomes, determining optimal cut-off values. Logistic regression analyzed SAA's association with clinical outcomes, adjusting for confounders. Kaplan-Meier analysis compared survival rates between high and low SAA groups, with log-rank tests for significance. Cox regression assessed SAA's independent prognostic value. NRI and IDI calculations evaluated SAA's added value to existing prognostic scores. $P<0.05$ was considered significant, with Bonferroni correction applied for multiple comparisons where appropriate.

Ethical Considerations

Before initiating the research, the University Medical Center's Institutional Review Board examined and endorsed the study protocol. The investigation adhered to the ethical standards outlined in the Declaration of Helsinki and followed Good Clinical Practice guidelines throughout its execution.

RESULTS

Table 1: Baseline Characteristics of Study Participants

Characteristic	All Patients (n=200)	Survivors (n=172)	Non-survivors (n=28)	P-value
Age, years (mean ± SD)	65.3 ± 17.2	63.1 ± 16.8	78.4 ± 12.9	<0.001
Male sex, n (%)	112 (56.0)	94 (54.7)	18 (64.3)	0.346
Comorbidities, n (%)				
- Chronic heart disease	58 (29.0)	46 (26.7)	12 (42.9)	0.082
- COPD	42 (21.0)	34 (19.8)	8 (28.6)	0.289
- Diabetes mellitus	36 (18.0)	28 (16.3)	8 (28.6)	0.115
PSI score (mean ± SD)	92.6 ± 35.7	86.9 ± 32.4	128.7 ± 31.5	<0.001
CURB-65 score (median [IQR])	2 [1-3]	2 [1-2]	3 [2-4]	<0.001

Table 2: Comparison of Biomarker Levels Between Survivors and Non-survivors

Biomarker	Survivors (n=172)	Non-survivors (n=28)	P-value
SAA, mg/L (median [IQR])	142.5 [68.3-287.6]	389.7 [210.5-612.8]	<0.001
CRP, mg/L (median [IQR])	98.5 [45.2-176.3]	187.3 [112.6-298.5]	<0.001
PCT, ng/mL (median [IQR])	0.48 [0.15-2.34]	2.87 [0.95-8.62]	<0.001
WBC, ×10^9/L (mean ± SD)	12.8 ± 5.7	15.9 ± 7.2	0.008

Table 3: ROC Analysis for Prediction of 30-day Mortality

Biomarker	AUC (95% CI)	Optimal Cut-off	Sensitivity	Specificity	PPV	NPV
SAA	0.86 (0.79-0.93)	275 mg/L	82.1%	79.1%	38.3%	96.5%
CRP	0.78 (0.70-0.86)	150 mg/L	75.0%	72.1%	29.2%	95.1%
PCT	0.81 (0.73-0.89)	1.5 ng/mL	78.6%	75.6%	33.3%	95.6%

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Table 4: Multivariate Logistic Regression Analysis for 30-day Mortality

Variable	Adjusted OR (95% CI)	P-value
Age (per year increase)	1.05 (1.02-1.08)	0.001
PSI score (per point increase)	1.02 (1.01-1.03)	0.002
SAA >275 mg/L	3.85 (1.68-8.82)	0.001
CRP >150 mg/L	1.92 (0.84-4.39)	0.122
PCT >1.5 ng/mL	2.31 (1.03-5.18)	0.042

Table 5: Net Reclassification Improvement (NRI) and Integrated Discrimination Improvement (IDI) for Adding SAA to Existing Prognostic Models

Model	NRI (95% CI)	P-value	IDI (95% CI)	P-value
PSI + SAA vs. PSI alone	0.21 (0.09-0.33)	0.001	0.056 (0.024-0.088)	<0.001
CURB-65 + SAA vs. CURB-65 alone	0.18 (0.06-0.30)	0.003	0.048 (0.018-0.078)	0.002

DISCUSSION

The results of our study provide compelling evidence for the potential utility of Serum Amyloid A (SAA) as a prognostic marker in community-acquired pneumonia (CAP). Our findings demonstrate that SAA levels are significantly elevated in patients with poor outcomes, particularly those who did not survive within the 30-day follow-up period. Furthermore, SAA showed superior predictive performance compared to traditional biomarkers and added incremental value to existing prognostic scores.

As shown in Table 1, our study included 200 patients with CAP, of whom 28 (14%) did not survive within 30 days of admission. This mortality rate is consistent with previous large-scale studies on CAP, such as the meta-analysis by Fine et al. (2016), which reported a pooled 30-day mortality rate of 13.7% across 23 studies. The significant differences in age, PSI scores, and CURB-65 scores between survivors and non-survivors align with established risk factors for poor outcomes in CAP (Lim et al., 2003; Fine et al., 1997).

Our results (Table 2) demonstrate that SAA levels were significantly higher in non-survivors compared to survivors (median 389.7 mg/L vs. 142.5 mg/L, $p<0.001$). This marked elevation in SAA levels among patients with poor outcomes is consistent with the findings of Nakayama et al. (2016), who reported a strong association between SAA levels and disease severity in CAP patients. Interestingly, while C-reactive protein (CRP) and procalcitonin (PCT) also showed significant differences between survivors and non-survivors, the magnitude of elevation for SAA appeared more pronounced. This observation supports the hypothesis that SAA might offer enhanced sensitivity in reflecting the severity of the inflammatory response in CAP, as suggested by Malle and De Beer (1996) in their review of SAA as an acute-phase reactant.

The ROC analysis (Table 3) revealed that SAA demonstrated excellent discriminatory power for predicting 30-day mortality, with an AUC of 0.86 (95% CI: 0.79-0.93). This performance surpassed that of CRP (AUC 0.78) and was slightly superior to PCT (AUC 0.81). The optimal cut-off value of 275 mg/L for SAA yielded a sensitivity of 82.1% and specificity of 79.1% for predicting mortality. These findings are particularly noteworthy when compared to previous studies on biomarkers in CAP. For instance, Chalmers et al. (2018) reported an AUC of 0.80 for PCT in predicting 30-day mortality, while Kruger et al. (2010) found an AUC of 0.76 for CRP. Our results suggest that SAA may offer improved prognostic accuracy over these established biomarkers. The high negative predictive value (NPV) of 96.5% for SAA at the optimal cut-off is especially relevant from a clinical perspective. This high NPV indicates that SAA could be particularly

useful in identifying low-risk patients who might be suitable for outpatient management, potentially reducing unnecessary hospitalizations and healthcare costs.

The multivariate logistic regression analysis (Table 4) confirmed the independent prognostic value of SAA after adjusting for age, disease severity (PSI score), and other biomarkers. An SAA level above 275 mg/L was associated with a nearly four-fold increase in the odds of 30-day mortality (adjusted OR 3.85, 95% CI: 1.68-8.82, p=0.001). This independent association remained significant even after accounting for established prognostic factors and other biomarkers. These findings are in line with the work of Tosanguan and Kitiyakara (2018), who reported that elevated SAA levels on admission were independently associated with adverse outcomes in CAP patients. However, our study extends these observations by demonstrating the superiority of SAA over CRP and its complementary value to PCT in a multivariate model.

One of the most significant findings of our study is the added prognostic value of SAA when combined with existing severity scores. As shown in Table 5, the addition of SAA to both the PSI and CURB-65 scores resulted in significant improvements in risk reclassification (NRI) and integrated discrimination (IDI). The NRI of 0.21 for PSI+SAA versus PSI alone indicates that 21% of patients were correctly reclassified in terms of their mortality risk when SAA was added to the model. Similarly, the IDI of 0.056 suggests a meaningful improvement in the model's ability to distinguish between survivors and non-survivors. These improvements were also observed, albeit to a slightly lesser extent, when SAA was added to the CURB-65 score. These results are particularly important in the context of previous attempts to improve upon existing prognostic scores. For example, Schuetz et al. (2017) reported an NRI of 0.11 when adding PCT to the PSI score, which is lower than the improvement we observed with SAA. Our findings suggest that SAA could be a valuable addition to current risk stratification tools, potentially enhancing their accuracy and clinical utility.

The strong prognostic performance of SAA in our study may be explained by its unique biological properties and functions in the acute-phase response. Unlike CRP, which is primarily produced in response to IL-6, SAA synthesis is induced by a broader range of cytokines, including IL-1, TNF-α, and IL-6 (Uhlar and Whitehead, 1999). This more comprehensive reflection of the inflammatory milieu may account for its enhanced prognostic accuracy. Furthermore, SAA has been shown to play active roles in the modulation of innate immunity, including the recruitment of immune cells and the induction of cytokine and chemokine production (Eklund et al., 2012). These functions suggest that SAA levels may not only reflect the intensity of the inflammatory response but also indicate the quality and effectiveness of the host defense against pulmonary pathogens.

The findings of our study have several important clinical implications. First, the superior prognostic performance of SAA suggests that it could be a valuable addition to the initial assessment of patients with CAP. Its high sensitivity and NPV make it particularly useful for identifying low-risk patients who might be safely managed in an outpatient setting, potentially reducing healthcare costs and the risk of hospital-acquired complications. Second, the independent prognostic value of SAA, even after adjusting for established severity scores, indicates that it provides additional information beyond traditional clinical and laboratory parameters. This suggests that SAA could be used to fine-tune risk stratification, especially in cases where clinical judgment and existing scores provide conflicting information. Third, the significant improvement in prognostic accuracy when SAA is added to PSI and CURB-65 scores opens the possibility of developing enhanced risk stratification tools that incorporate this biomarker. Such refined tools could lead to more personalized management strategies for CAP patients, aligning with the growing trend towards precision medicine in infectious diseases.

Limitations and Future Directions

Despite the promising results, our study has several limitations that should be acknowledged. First, as a single-center study with a relatively small sample size, our findings require validation in larger, multi-center cohorts. Second, we did not assess the

kinetics of SAA levels over time, which could provide additional prognostic information. Future studies should consider serial measurements to evaluate the prognostic value of SAA trends during the course of illness.

Additionally, while we focused on 30-day mortality as the primary outcome, future research should explore the relationship between SAA levels and other important clinical outcomes, such as need for mechanical ventilation, length of hospital stay, and long-term functional status. It would also be valuable to investigate whether SAA-guided management strategies could improve patient outcomes compared to standard care. Furthermore, the potential role of SAA in differentiating between bacterial and viral etiologies of CAP, as well as its utility in guiding antibiotic therapy, remains to be explored. Studies comparing SAA with other emerging biomarkers, such as pro-adrenomedullin or soluble triggering receptor expressed on myeloid cells-1 (sTREM-1), would also be informative in defining its relative strengths and potential synergies.

CONCLUSION

Our study provides strong evidence for the prognostic value of SAA in patients with CAP. Its superior performance compared to traditional biomarkers and its ability to enhance existing severity scores suggest that SAA could be a valuable addition to the clinical assessment of CAP patients. Further research is warranted to validate these findings in diverse populations and to explore the potential of SAA-guided management strategies in improving outcomes for patients with this common but potentially severe infection.

CONFLICTS OF INTEREST

None

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UTHORS CONTRIBUTION

All authors have equal contribution.

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None

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